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DEFENSE MECHANISMS AND DISSOCIATION AS MEDIATORS OF THE ASSOCIATION BETWEEN POLYTRAUMATIZATION AND SOMATIZATION IN FAROESE AND INDIAN ADOLESCENTS

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Dissertação apresentada à Faculdade de Ciências Sociais e Tecnologia da Universidade Europeia, para cumprimento dos requisitos necessários à obtenção do grau de Mestre em *Psicologia Clínica e da Saúde* realizada sob a orientação científica do Doutor Paulo Alexandre da Silva Ferrajão, *Professor Auxiliar da Universidade Europeia* e coorientação do Doutor Ask Elklit, *Professor da Universidade do Sul da Dinamarca*

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Resumo

A exposição a múltiplas experiências adversas na infância (ACE) é um fator de risco para o desenvolvimento e aumento de sintomas de somatização em adolescentes. Os mecanismos de defesa e a dissociação podem desempenhar um papel fundamental na associação entre essas variáveis. O objetivo da presente dissertação é analisar a associação entre a exposição direta e indireta a ACE e os sintomas de somatização em adolescentes das Ilhas Faroé e da Índia, e explorar o papel mediador dos mecanismos de defesa e dos sintomas de dissociação nesta associação. A amostra foi constituída por 687 adolescentes das Ilhas Faroé e 411 adolescentes indianos, que completaram questionários de autorrelato validados que avaliaram as variáveis do estudo. Os dados foram analisados através do teste de modelos de equações estruturais efetuando-se o teste de modelos múltiplos em série segundo os procedimentos de Preacher e Hayes (2008). Os resultados indicaram que a associação entre as variáveis é diferente para adolescentes das Ilhas Faroé e da Índia. Os sintomas de dissociação mediaram a associação entre a exposição direta e indireta a ACE e os sintomas de somatização em ambas as amostras. Na amostra das Ilhas Faroé, os mecanismos de defesa maduros foram mediadores da associação entre a exposição indireta a ACE e os sintomas de somatização. Os modelos múltiplos em série indicaram que maior exposição direta e indireta a ACE estava significativamente associada a níveis mais elevados de mecanismos de defesa imaturos, que estavam associados a níveis mais elevados de sintomas de dissociação e que, por sua vez, estavam associados a níveis mais elevados de sintomas de somatização. Na amostra indiana, os mecanismos de defesa não mediaram a associação entre a exposição direta e indireta a ACE e os sintomas de somatização. Os modelos múltiplos em série indicaram que a exposição direta a ACE estava significativamente associada a níveis mais elevados de mecanismos de defesa imaturos, que estavam associados a níveis mais elevados de sintomas de dissociação e que, por sua vez, estavam associados a níveis mais elevados de sintomas de somatização. Os resultados obtidos na presente dissertação sugerem que níveis elevados de sintomas de dissociação e mecanismos de defesa imaturos, bem como níveis mais baixos de mecanismos de defesa maduros, podem ser considerados fatores de vulnerabilidade no agravamento dos sintomas de somatização.

Palavras-chave: experiências adversas na infância; adolescência; mecanismos de defesa; dissociação; somatização

Abstract

Exposure to multiple adverse childhood experiences (ACE) is a risk factor for the development and increase in somatization symptoms among adolescents. Defense mechanisms and dissociation may play a key role in the association between these variables. Our aim was to analyze the associations between direct and indirect exposure to ACE and somatization symptoms in Faroese and Indian adolescents and to explore the mediating role of defense mechanisms and dissociation symptoms in this association. The sample consisted of 687 Faroese adolescents and 411 Indian adolescents, who completed validated self-report questionnaires that assessed the variables under study. Serial multiple mediation models were tested by conducting a structural equation modeling employing Preacher and Hayes' procedures (2008). Results showed that the association between these variables is different for Faroese and Indian adolescents. Dissociation symptoms mediated the association between direct and indirect exposure to ACE and somatization symptoms in both samples. In the Faroese sample, mature defense mechanisms were mediators of the association between indirect exposure to ACE and somatization symptoms. Multiple serial models indicated that higher direct and indirect exposure to ACE were significantly associated with higher levels of immature defense mechanisms, which were associated with higher levels of dissociation symptoms, which in turn were associated with higher levels of somatization symptoms. In the Indian sample, defense mechanisms did not mediate the association between direct and indirect exposure to ACE and somatization symptoms. Multiple serial models indicated that direct exposure to ACE was significantly associated with higher levels of immature defense mechanisms, which were associated with higher levels of dissociation symptoms, which in turn were associated with higher levels of somatization symptoms. The results obtained in the present dissertation suggest that high levels of dissociation symptoms and immature defense mechanisms and lower levels of mature defense mechanisms might be seen as vulnerability factors, in aggravating somatization symptoms severity.

Keywords: adverse childhood experiences; adolescence; defense mechanisms; dissociation; somatization

Index

List of Tables and Figures

Introduction

Polytraumatization, that is, multiple exposure to distinct traumatic experiences, is pervasive during younger years and may contribute significantly to mental and physical health problems among youth (Bomysoad & Francis, 2020; Finkelhor et al., 2015a; Hughes et al., 2017). Although most research about polytraumatization and its effects has been conducted in high and upper-middle-income countries, low- and middle-income countries (LALMIC) are home to most of the world's children and adolescents, who face a much higher risk of being exposed to multiple forms of adverse childhood experiences (ACE) (Le et al., 2018; Pereda et al., 2014). In the Faroe Islands, an autonomous territory within the Kingdom of Denmark and a high income country, adolescents have reported high rates of exposure to adverse experiences, which can be due to the country's particular context, namely, being a family-centered society reliant on high mobility jobs, encouraging of early autonomy and independence and lacking in parental supervision, as well as having faced economic stressors (Gaini, 2013; Gaini & Sleire, 2023; Hangstova Føroya, 2022; Hayfield, 2018; Petersen et al., 2010). In comparison, India is a LALMIC where economic precarity, poor living conditions and violence are common, leading adolescents to be exposed to different types of traumatic events consistently (Das et al., 2012; Fernandes et al., 2021; Rasmussen et al., 2013). Despite that, polytraumatization research in these countries is lacking, especially comparing such distinct socioeconomic and cultural contexts.

A well-documented and common consequence of polytraumatization is somatic symptoms (Kugler et al., 2012; Schulte & Petermann, 2011). However, the findings concerning their association have not been unanimous, leading some authors to propose that other variables may influence this relationship (Guerra et al., 2016). For instance, defense mechanisms and dissociation have been identified as processes that play an important role in the psychological adjustment to trauma (Luoni et al., 2018; Punamäki et al., 2002). Dissociation has been proposed as a coping response, through which the activation of altered states of consciousness acts as a protection against traumatic experiences (Gullestad, 2005). Dissociation has also been associated with somatic symptoms and is suggested that trauma is a common etiological factor that links both (van der Kolk et al., 1996). Moreover, the core functions of defense mechanisms in the aftermath of trauma involve the implicit regulation of emotions, influencing how individuals deal

with memories and feelings and aiming to hide or alleviate conflicts or stressors that cause anxiety to exacerbate (Beresford, 2012). Nonetheless, reliance on immature defense mechanisms has been associated with increased somatic symptom severity (Hyphantis et al., 2013a).

Thus, there is evidence that the aforementioned variables (polytraumatization, defense mechanisms, dissociation and somatization symptoms) are associated and, to our knowledge, no other study has analyzed these variables together in the same study. Considering the existing gaps in research on Faroese and Indian adolescents, it is relevant to analyze the role of defense mechanisms and dissociation as mediators of the association between exposure to ACE and somatization symptoms, which we aim to do in this dissertation. Its main goals are to analyze the association between direct and indirect exposure to ACE and somatization symptoms; to assess the mediating role of both defense mechanisms and dissociation symptoms in the relationship between direct and indirect exposure to multiple ACE; and to examine the series of associations between exposure to multiple ACE, defense mechanisms, dissociation symptoms and somatization symptoms. The present dissertation contains an Empirical Article, where the association between the variables under study was analyzed, results were discussed according to the existing literature, and the main conclusions were drawn. Study limitations are also addressed. Finally, an overall discussion is presented, where results are subjected to more in-depth analysis, clinical implications are explained, and recommendations for future studies are provided.

Empirical Article

Abstract

Exposure to multiple adverse childhood experiences (ACE) is a risk factor for the development and increase in somatization symptoms among adolescents. Defense mechanisms and dissociation may play a key role in the association between these variables. Our aim was to analyze the associations between direct and indirect exposure to ACE and somatization symptoms in Faroese and Indian adolescents and to explore the mediating role of defense mechanisms and dissociation symptoms in this association. The sample consisted of 687 Faroese adolescents and 411 Indian adolescents, who completed validated self-report questionnaires that assessed the variables under study. Serial multiple mediation models were tested by conducting a structural equation modeling employing Preacher and Hayes' procedures (2008). Results showed that the association between these variables is different for Faroese and Indian adolescents. Dissociation symptoms mediated the association between direct and indirect exposure to ACE and somatization symptoms in both samples. In the Faroese sample, mature defense mechanisms were mediators of the association between indirect exposure to ACE and somatization symptoms. Multiple serial models indicated that higher direct and indirect exposure to ACE were significantly associated with higher levels of immature defense mechanisms, which were associated with higher levels of dissociation symptoms, which in turn were associated with higher levels of somatization symptoms. In the Indian sample, defense mechanisms did not mediate the association between direct and indirect exposure to ACE and somatization symptoms. Multiple serial models indicated that direct exposure to ACE was significantly associated with higher levels of immature defense mechanisms, which were associated with higher levels of dissociation symptoms, which in turn were associated with higher levels of somatization symptoms. The results obtained in the present dissertation suggest that high levels of dissociation symptoms and immature defense mechanisms and lower levels of mature defense mechanisms might be seen as vulnerability factors, in aggravating somatization symptoms severity.

Keywords: adverse childhood experiences; adolescence; defense mechanisms; dissociation; somatization

Defense Mechanisms and Dissociation as Mediators of the Association between Polytraumatization and Somatization in Faroese and Indian Adolescents

Adverse Childhood Experiences (ACE) are conceptualized as potentially traumatic events that can take place during developmental years, prior to the age of 18 (Contractor et al., 2018). Such experiences can be broadly defined as multiple forms of abuse and neglect, as well as household dysfunctions (Felitti et al.,1998). Recent research has expanded on the concept, adding childhood adversities that occur outside the home, namely bullying, exposure to community and collective violence, and low socio-economic status (Cronholm et al., 2015; Finklehor et al., 2015b; Struck et al., 2021). Furthermore, one can be exposed to ACE directly or indirectly. Direct exposure consists of experiencing adversity firsthand, and indirect exposure refers to witnessing or learning about others' experience of adversity (Patias & Dell'Aglio, 2017). Both types of exposure have been associated with various forms of distress and maladjustment. However, there is some evidence that direct and indirect exposure to ACE have differential effects on individuals' mental health (Ferrajão et al. 2022).

It has been well established that the exposure to ACE is pervasive during younger years (Finkelhor et al., 2015a). For example, most of U.S adolescents (60%) reported experiencing at least one traumatic event (Vacek & Whisman, 2021). Some authors have indicated that adolescents present increased vulnerability to being exposed to ACE (Breslau et al., 2004; Finklehor et al., 2007; Nooner et al., 2012). Adolescence is viewed as a sensitive period of development, when biological and psychological developmental changes, as well as increasingly complex social challenges take place. Adolescents face demands such as developing more independence from their parents, establishing relations with peers, and achieving a coherent sense of self (Cicchetti & Rogosch, 2002; Erickson, 1982). It is during adolescence that individuals go through new experiences, related to the developmental tasks specific to this period, often engaging in risk-taking behavior such as drinking alcohol, drug use and sexual-risk behaviors, which tend to co-occur (Leather, 2009). These risky behaviors can put adolescents at a greater risk for exposure to multiple ACE (Champion et al., 2004; Maniglio, 2017). In turn, being exposed to ACE in this period can sabotage healthy adjustment and have negative effects on health and well-being throughout one's life course (Bomysoad & Francis, 2020; Hughes et al., 2017; Meeker et al., 2021).

Previous research has focused almost entirely on the effects of a single adverse experience on mental health. An unrecognized aspect of seminal literature concerning ACE, is the interrelation and cumulative burden that such experiences tend to have (Dong et al., 2004; Finklehor et al., 2007). In this sense, research has indicated that children and adolescents exposed to one traumatic experience are more likely to experience further trauma (Wolfe, 2018). Moreover, multiple exposure to distinct traumatic experiences, that is polytraumatization, has been identified to be highly prevalent among youth, and, compared to a single or repeated experience of a specific type of trauma, is a stronger predictor of mental and physical health problems, including somatization, in children and adolescents (Álvarez-Lister et al., 2014; Ford et al., 2010; Gustafsson et al., 2009).

In literature, the concept of polytraumatization has been operationalized in three different ways: the categorical approach posits that individuals can be classified as being polytraumatized or not by using a predefined criteria, such as having been exposed to more types of traumatic experiences than average for a given sample (Finklehor et al., 2007); the hierarchical approach suggests that some types/combinations of traumatic experiences, such as sexual abuse, are inherently more detrimental than others (Lau et al., 2005); and the cumulative approach, which we chose to study, proposes that the number of traumatic experiences an individual is exposed determines outcomes in a dose-response manner, such that exposure to multiple traumatic experiences result in poorer outcomes (Adams et al., 2016).

Research pertaining to polytraumatization among adolescents has been primarily conducted in North America and northern Europe (Pereda et al., 2014). Some studies in northern European countries have found a prevalence of polytraumatization among adolescents that ranged between 8 and 10%, using a categorical operationalization (Aho et al., 2014; Ellonen & Salmi, 2011; Mossige & Huang, 2017). For example, in the Faroe Islands, Petersen et al. (2010) found that 90% of adolescents reported to have witnessed or directly experienced at least one traumatic event. These findings may be associated with factors particular to the Faroe Islands context.

With a current population of approximately 54,000 people, the Faroe Islands are an autonomous region within the Danish Kingdom, and a high-income country with high standards of living (Hangstova Føroya, 2022; Petersen et al., 2010). The Faroe Islands are heavily

dependent on fisheries, which historically has led men working at sea to be absent from home for long periods of time. This is still a reality in small village communities and, although in more urbanized areas parents are more present, many modern jobs require high mobility (Hayfield, 2018). As a family-centered society, where the father symbolizes the main link between the nuclear family and the society, young people feel vulnerable in the absence of the father (Gaini, 2013; Gaini & Sleire, 2023). Besides family, the Faroese also value autonomy and independence, thus youth grow up with little guidance from their parents. From an early age, children are allowed to play unsupervised, and are expected to solve problems on their own or to rely on their peers (Gaini, 2013). Furthermore, small-scale societies such as the Faroe Islands, are characterized by close social networks and high levels of familiarity. Although this means people identify more deeply with one another, it also means that anonymity is almost impossible (Hayfield & Schug, 2019). Moreover, the Faroe Islands also represent a society facing the challenges of economic restructuration and out-migration, due to the economic crisis in the 1990s (Gaini & Sleire, 2023).

Contrastingly, low- and middle-income countries (LALMIC) are home to most of the world's children and adolescents. About 38% of youth living in LALMIC have reported polytraumatization, which is a higher prevalence than in high- and upper-middle income countries (Le et al., 2018). Children and adolescents who live in LALMIC also face a much higher risk of being exposed to multiple forms of adversity compared to those living in high- and upper-middle income countries. Childhood adversity in these countries can be fueled by poverty, war, low educational levels, poor health care systems, prevalence of diseases such as HIV/AIDS, but also cultural beliefs and practices (Benjet, 2010; Le et al., 2018). It should be noted that family structure also seems to be related with ACE and polytraumatization on a universal level. Previous studies have found that the risk of exposure to ACE is significantly higher among youth living with single parents and stepparents compared to those living with both biological parents (Berger, 2004; Turner et al., 2013; McChesney et al., 2015). Finklehor et al. (2005) also found that children and adolescents who suffered polytraumatization were more likely to be from single-parent families.

India is considered a LALMIC, with a population as of the last Census of 1.2 billion people (Government of India, n.d.). In addition to its large population, India is characterized by

its diversity of cultures, languages, ethnic groups, and religions (Avasthi et al., 2013). Unlike western societies, India is a collectivist society that promotes interdependence, with the family unit serving as the cornerstone of this social framework (Chadda & Deb, 2013). Indian families share close ties and family members rely on each other for emotional support, social interaction, and financial assistance (Hsieh et al., 2023). The family is considered the fundamental source of growth and development for Indian children, and therefore, it has a vital responsibility in safeguarding and providing them with support (Sooryamoorthy, 2012). Nonetheless, India has a unique and complex socioeconomic and cultural context that constitutes a risk to the experience of adversity (Saraswathi & Oke, 2013). The Indian context is marked by extreme poverty, poor healthcare, cultural beliefs in harsh discipline, gender-based inequalities and violence, terrorism and political conflict, as well as natural disasters (Das et al., 2012; Deb et al., 2016a; Rasmussen et al., 2013). Existing studies indicate that Indian adolescents are often exposed to different types of adversity (Fernandes et al., 2021; Maurya & Maurya, 2023). A study with youth from Pune City found that 78.1% of the sample experienced at least one potentially traumatizing event, directly or indirectly (Rasmussen et al., 2013).

There is still a dearth of studies on polytraumatization and its effects in these two countries. Nonetheless, given the differences in their socioeconomic and cultural context, adolescents living in the Faroe Islands and in India may have distinct experiences of exposure to ACE. Furthermore, as previously mentioned, these experiences can have a deleterious effect on adolescents' mental and physical health. Considering that somatization is a common consequence of polytraumatization and involves both mental and physical distress, it is our aim to study the association between polytraumatization and somatization in adolescents from both countries.

Somatization is defined as the experience of physical symptoms, which may or may not be explained by a medical condition, as well as abnormal thoughts, feelings, and behaviors associated with these symptoms, that cause significant distress (American Psychiatric Association, 2013). Somatic symptoms can manifest in different ways, but common complaints in the pediatric population include fatigue, dizziness, headaches and abdominal pain (Bonvanie et al., 2017). Although somatic symptoms are not unusual among youth, they seem to be extremely prevalent among children and adolescents exposed to traumatic experiences (Kugler et

al., 2012; Schulte & Petermann, 2011). In fact, Charak et al. (2019) found that adolescents who suffered polytraumatization reported higher levels of somatic complaints compared to those not exposed to trauma or exposed to a singular traumatic event. In addition, a study with adolescent outpatients found that those who suffered polytraumatization were five times more susceptible to present clinically significant somatic symptoms than adolescents who were exposed to one specific type of trauma (Álvarez-Lister et al., 2014). In this respect, Ford et al. (2013) observed a positive association between polytraumatization and somatic complaints among justice-involved adolescents. Likewise, Pinto et al. (2022) demonstrated that cumulative adversity had a direct and a large effect size on somatization in a study with adolescents living in high-risk contexts.

Nonetheless, research has not been unanimous in its findings concerning the association between polytraumatization and somatization. A study with child and adolescent outpatients in psychiatric treatment revealed that polytraumatization was not associated with a measure that assessed somatic complaints (Ford et. al, 2011). In this regard, it has been proposed that different variables may play a mediating role in the relationship between polytraumatization and somatization (Guerra et al., 2016). Therefore, this relationship remains unclear, which may be due to underlying psychological mechanisms that have not been sufficiently investigated. In this study we suggest that dissociation and defense mechanisms might mediate this relationship.

An early explanation of dissociation was offered by Janet (1889). The author defined dissociation as a pathological separation between ideas, behaviors and consciousness, with which the individual reacts to trauma. In this view, due to the frightening nature of traumatic experiences, these could not be successfully integrated into the memory system and would then split off from consciousness (Van der Kolk & Van der Hart, 1989). Contemporary studies of dissociation have been aligned with Janet´s view of dissociation as a failure of synthesis and integration, which implies a disturbance in the cognitive processing of traumatic memories (Gullestad, 2005; Scalabrini et al., 2020). This view was also captured by the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) (APA, 2013) which describes dissociation as a "disruption of and/or discontinuity in the normal integration of consciousness, memory, identity, emotion, perception, body representation, motor control and behavior" (APA, 2013, p. 291). Furthermore, dissociation can manifest in different ways: as negative symptoms and as positive symptoms. Negative symptoms refer to the inability to access information or check mental

functions that are generally accessible or controllable, such as amnesia. Positive symptoms include trauma-related intrusions that invade consciousness and behavior, associated with the loss of continuity of subjective experience, such as fragmentation of identity and depersonalization (Luoni et al., 2018).

Experiencing dissociative symptoms is more frequent in individuals exposed to multiple and repetitive traumatic events (Luoni et al., 2018). In fact, Nilsson et al. (2010) observed that polytraumatization contributed significantly to the prediction of symptoms of dissociation among adolescents. In this perspective, some studies support the idea that dissociative symptoms may be a coping response to deal with traumatic experiences, since these experiences can be made less intense through dissociative alterations in perception and allow the person to forget or even disown the experience as occurring in another person (Chu & Dill, 1990; Nijenhuis et al., 1998). Dissociation can thus be seen as a basic part of the trauma response, implying a protective activation of altered states of consciousness in reaction to overwhelming painful events (Gullestad, 2005). However, it is known that dissociation often remains as a way of coping with subsequent stress, causing people to become emotionally constricted and even develop psychopathology (van der Kolk et al., 1996).

Moreover, dissociation can make memories of events unavailable or only partially available (Egeland & Susman-Stillman, 1996). In some people, the memories of traumatic events may be entirely organized on an implicit or perceptual level and have no verbal component, which interferes with the disclosure of the traumatic experience. It has been suggested that trauma is organized in memory on sensory-motor and affective levels. Thus, the memory of the traumatic experience in individual's who dissociate can consist in fragmentary perceptual components such as visual images, smells, sounds, somatosensory sensations or intense waves of feelings (Van der Kolk & Fisler, 1995).

Dissociation has been associated with somatic symptoms. For example, Farley and Keaney (1997) observed that dissociation and somatization were significantly positively associated in women with a history of childhood sexual abuse. Additionally, Luoni et al. (2018) found that adolescents with a history of exposure to multiple traumatic experiences and dissociative symptoms presented higher rates of somatization. Furthermore, there is evidence that dissociation may mediate the association between exposure to ACE and somatization. For

instance, Salmon et al. (2003) observed that individuals who present a history of exposure to traumatic experiences reported greater dissociation symptoms which, in turn, were associated with a tendency to report somatic symptoms. These findings suggest that traumatic memories may be dissociated from consciousness and later manifest as somatic symptoms (Saxe et al., 1994). It can also be proposed that trauma is a common etiological factor that links dissociation and somatization (van der Kolk et al., 1996).

Furthermore, there is indication that the relationship between ACE exposure and dissociation may be mediated by other psychological processes. Dissociative symptoms might be influenced by emotion regulation difficulties in the face of trauma and its consequences (Chaplo et al., 2015; Powers et al., 2015). In this regard, the most addressed form of emotion regulation is explicit emotion regulation, such as coping skills, which involves effortful conscious cognitive manipulations of emotional responses. Nonetheless, emotion regulation also includes implicit emotion regulation, that is, automatic processes aimed to modify the quality, intensity, or duration of an emotional response, which operate outside of conscious awareness (Koole & Rothermund, 2011; Rice & Hoffman, 2014). There is growing recognition of implicit emotion regulation as vital components of self-regulation and mental health. These seem to be more important than explicit emotion regulation, since they do not require conscious effort or monitoring (Gyurak et al. 2011). Implicit processes also seem to facilitate efficient mind-body interactions that are associated with effective emotion regulation (Koole & Rothermund, 2011).

Implicit emotion regulation is inherently linked to defense mechanisms (Rice & Hoffman, 2014). Like implicit emotion regulation, defense mechanisms operate at an unconscious level aiming to protect the individual from anxiety and other unpleasant emotions, also playing an important role for successful psychological adaptation (Lee et al., 2021; Prout et al., 2019). The concept of defense mechanism was first introduced by Sigmund Freud. Defense mechanisms were considered a general designation for all the techniques which the ego utilizes in intrapsychic conflicts that potentially lead to neurosis (Freud,1926). In this light, defenses would develop to modify, distort, or avoid disturbing thoughts, feelings, and perceptions, since its recognition would create anxiety. Defenses were initially linked to psychopathology, however, Anna Freud (1936) reintroduced the use of defenses as part of normal psychological functioning and development (Cramer, 2015).

Although they are no longer contemplated in the Diagnostic and Statistical Manual of Mental Disorders 5 (DSM-5; APA, 2013), defense mechanisms are a central concept in psychoanalytic psychotherapy, widely recognized for its clinical utility (Vaillant, 2011). Currently, defense mechanisms can be understood as unconscious and automatic psychological processes that mediate individuals' responses to internal and external stressors. Their aim is to maintain individuals' psychological stability and prevent awareness of these stressors (Perry, 2014; Vaillant, 1994). Vaillant (1986) proposed a developmental hierarchy of defense mechanisms, organized through levels of maturity and adaptiveness into three levels. First, immature or maladaptive defenses (e.g., projection, splitting), which protect individuals from unpleasant experiences or conflicts by constricting awareness of ideas, feelings, and actions. Second, intermediate or neurotic defenses (e.g. idealization, repression) that keep potentially threatening memories, ideas, feelings or fears out of awareness. And finally, mature or adaptive defenses (e.g., humor, sublimation) which maximize gratification and reinstate psychological homeostasis, while allowing more conscious awareness of stressors (Perry et al., 2015; Vaillant, 2011).

Defense mechanisms begin to develop in infancy and continue to develop as personality matures (Vaillant, 2011). Some defense mechanisms are more prominent in specific stages of the development. In this sense, studies have demonstrated that immature defenses are predominant in childhood and are later replaced by more mature defenses in adolescence (Cramer, 2018; Porcerelli et al., 1998). Moreover, Di Giuseppe et al. (2019) found that younger adolescents were more likely to use immature defense mechanisms compared to older teens, who instead tended to use neurotic and mature defenses more. Adolescents who rely on immature defenses seem to exhibit higher levels of maladjustment, while adolescents who make use of mature defenses express higher levels of perceived competence (Sandstorm & Cramer, 2003).

Following traumatic experiences, defense mechanisms aim to hide or alleviate conflicts or stressors that cause anxiety to exacerbate (Beresford, 2012). Therefore, defenses can be seen as protective psychological processes, decisive in determining adjustment to trauma (Punamäki et al., 2002). However, traumatic experiences can alter mental processes, specifically impacting defensive functioning (Perry et al., 2015). Thus, high exposure to such experiences can cause regression of mature defenses (Punamäki et al., 2002). In this sense, several studies have found a

positive association between childhood trauma and the use of immature and neurotic defense mechanisms, as well as a negative association between childhood trauma and mature defenses (Korkmaz et al., 2020; Romans et al., 1999; Uygur et al., 2022).

Previous studies analyzed the association between defense mechanisms and somatic symptoms. It was found that predominant use of immature defenses increased somatic symptom severity and that predominant use of mature defenses was associated with lower levels of somatic symptoms (Hyphantis et al., 2013a; Hyphantis et al., 2013b). Other studies have analyzed this association in individuals exposed to traumatic experiences, reporting similar findings. In a study with young adults attending psychotherapy with a history of childhood trauma, it was found that mature defense mechanisms were negatively associated with somatization and that neurotic and immature defenses were positively associated with somatization (Waikamp et al., 2021). Another study with adult inpatients who were exposed to childhood adversity demonstrated a positive and significant association between immature defenses and somatization, as well as the extent of the somatic complaints (Nickel & Eagle, 2006).

There are also some studies that have found associations between defense mechanisms and dissociative symptoms. It was observed that both neurotic and immature defenses were positively associated with dissociation and immature defenses were related to dissociation severity (Muris & Merckelbach, 1997; Simeon et al., 2003). Furthermore, there is also evidence linking defense mechanisms and dissociative symptoms in the context of trauma. In a study with women who suffered from childhood sexual abuse, dissociation was negatively associated with mature defenses (Romans et al., 1999).

Past research has also investigated the mediating role of defense mechanisms in the association between childhood trauma and somatization. For example, Finzi-Dottan and Karu (2006) found that immature defenses mediated the association between childhood emotional abuse and controlling parental behaviors and somatization in samples of adult students. Also, Nickel and Egle (2006) observed that childhood trauma and immature defense styles had an association with somatization, leading the authors to suggest that immature defense styles may act, in part, as mediators between these two variables. As previously mentioned, defense mechanisms play an important role in psychological adjustment to trauma, thus it can be

proposed that, contrary to mature defenses, immature and neurotic defenses seem to contribute significantly to maladjustment, since individuals exposed to traumatic experience who use such defenses manifest greater distress.

There is a lack of research on polytraumatization and its effects in the Faroe Islands and India. These two countries have unique socioeconomic and cultural contexts, which may contribute to adolescents having different levels of exposure to ACE. Given that somatization is a common consequence of polytraumatization, but has not yet been sufficiently studied in these populations, our first aim is to analyze whether the association between the direct and indirect exposure to ACE and somatization symptoms is different in the Faroe Islands and India. Furthermore, since the direct association between ACE and somatization has not always been consistent, in this study, we suggest defense mechanisms and dissociation as mediators of this relationship. As noted previously, exposure to ACE, defense mechanisms, dissociation, and somatization are all associated. However, there is a dearth of studies on the association between these variables on adolescents, specially from the Faroe Islands and India. To the best of our knowledge, no previous study examined the association between these variables in these populations. Therefore, our second aim is to analyze the effect of direct and indirect exposure to ACEs on somatization symptoms through the mediation of defense mechanisms and dissociation in Faroese and Indian adolescents.

Based on previous findings described above, the following hypothesis were proposed:

1. Country membership will moderate the link between exposure to ACE and somatization symptoms.
2. Higher direct and indirect exposure to ACE will be associated with higher levels of somatization symptoms.
3. Defense mechanisms will mediate the link between direct and indirect exposure to ACE and somatization symptoms.
4. Dissociation symptoms will mediate the link between direct and indirect exposure to ACEs and somatization symptoms.
5. Serial multiple mediation models will show that direct and indirect exposure to ACE will relate to higher levels of immature defense mechanisms and lower levels of

mature defense mechanisms, that will relate to higher levels of dissociation symptoms, which will then relate to higher levels of somatization symptoms.

Method

Participants

Sample characteristics are presented in Table 1.

Table 1

Sample Demographic Characteristics

	Faroese group (n= 687)	Indian Group (n = 411)	Combined sample (N = 1098)
Age			
13 years	20 (2.9%)	17 (4.1%)	37 (3.4%)
14 years	356 (51.8%)	316 (76.9%)	672 (61.2%)
15 years	304 (44.3%)	77 (18.7%)	381 (34.7%)
16 years	7 (1.0%)	1 (0.2%)	8 (0.7%)
Mean	14.4 (SD=0.6)	14.2 (SD =0.5)	16.1 (SD = 1.7)
Sex			
Male	326 (47.5%)	219 (53.3%)	519 (58.6%)
Female	361 (52.5%)	192 (46.7%)	366 (41.4%)
Living with			
Both parents	562 (81.8%)	395 (96.1%)	957 (87.2%)

One of their parents	111 (16.2%)	13 (3.2%)	124 (11.3%)
Other arrangements (uncles, siblings, grandparents or other relatives)	14 (2.0%)	3 (0.7%)	17 (1.5%)

There were no statistically significant differences in age between Faroese and Indian adolescents ($t(1096) = .01$, $p = .99$). Chi-square test for independence indicated no significant association between sex and sample, χ^2 (1, $n = 1098$) = 3.50, $p = .06$). Chi-square test for independence indicated significant association between people who they live and sample, χ^2 (4, $n = 1098$) = 47.33, $p < .001$). The proportion of adolescents who lived with one of their parents in the Faroese sample was higher compared with the Indian sample.

Procedure

The primary aim to conduct the study was to gather information on exposure to ACE and psychological reactions among adolescents living in different countries. These countries were selected because of the differences in their socioeconomical context. The Faroe Islands is an isolated, self-governing North Atlantic region, considered a high-income country with high social standards. In contrast, India is a LALMIC, overpopulated, with a unique and complex cultural context that constitutes a risk to the experience of adversity compared to Faroe Islands. The data were collected in 2012. Data collection was sponsored by the National Center of Psychotraumatology (Odense, Denmark).

Faroese adolescents were recruited from 19 schools located on six different islands that taught eighth grade students. According to the Faroese Ministry of Education, there were 804 students in eighth grade at the time the data were collected; that is 85% of all the Faroese students participated. Previous to data collection, all questionnaires and a letter explaining the objectives of the study, and the confidentiality procedures were sent to the Faroese Data Inspection, the Faroese Ministry of Education, and the Faroese Ethical Board, that approved the

study. Following, the same information was presented to the school principals which reviewed and approved the study. Finally, it was delivered to the ‘‘head teacher’’ (the teacher responsible for each class of students) information on the research protocol.

Students were introduced to information about the study aims, procedures and the role of the participant both verbally and by letter. The participation was voluntary and those accepting to participate, gave their informed consent directly. The students filled in the questionnaire in the classroom, supervised by a team researcher in co-operation with the “head teacher’’. The students were informed that their answers were anonymous and they were asked to answer as openly as possible. All students present accepted to participate in the study.

Indian adolescents who participated in the study were from the city of Pune in the state of Maharashtra. Due to limited resources such as time and finances, data collection was only conducted in Pune and only students from private schools were selected for the study. Before data collection, five schools were selected on a convenience basis and invitations to participate in the study were sent, but only two decided to enroll in the study. The students were primarily from a middle- and upper-class background. Each of the eight classes of students who participated in the study consisted of 50 to 60 adolescents. The headmasters and the boards of the schools reviewed and approved the study following the presentation of a questionnaire and a letter explaining the aims of the study.

It was conducted a pilot study with seven respondents at the age of 13-14 years. Information about the study aims, procedures and the role of the participant was introduced to the students verbally and by letter. The participation was voluntary and those accepting to participate, gave their informed consent directly. The questionnaires were filled by students in the classroom, supervised by a team researcher in co-operation with two Hindi speaking Indian psychology students, who explained the research protocol and practicalities in answering the questionnaire. The students were informed that their answers were anonymous, and they were asked to answer as openly as possible. All students present accepted to participate in the study.

Measures

Sociodemographic data. Participants provided information on their sex, age, and current living arrangements.

Potentially Traumatic Events. It was asked to participants if they had been exposed directly and/or indirectly to a list of 20 life-threatening experiences (e.g., rape) and stressful family conditions (e.g., neglect). The measure was developed by Bödvarsdóttir and Elklit (2007) who selected the list of events from scientific literature and clinical experience. This measure has been widely applied cross-culturally (e.g., Ferrajão & Elklit, 2021).

Defensive Style Questionnaire. The Defensive Style Questionnaire (DSQ-40; Andrews et al., 1993) assessed 20 defenses divided into 3 groups of factors: mature, neurotic, and immature. This measure includes 40 self-report questions answered on a 9-point Likert scale (where "1" indicates "completely disagree" and "9" indicates "fully agree"). In this study, the total scores on the mature, neurotic and immature defense mechanisms were analyzed. Cronbach alpha for the mature scale was .81 in the combined sample (Faroese =.82; Indian=.79), the neurotic scale was .80 in the combined sample (Faroese =.80; Indian=.80), and the immature scale was .80 in the combined sample (Faroese =.82; Indian=.75) was good.

Trauma Symptom Checklist. The Trauma Symptom Checklist (TSC-33; Briere & Runtz, 1989) was used to assess both dissociation and somatization symptoms. The TSC-33 includes 33 items rated with reference to the previous month. Items are answered on a 4-point Likert scale (from "never" = 1, to "very often" = 4). The sum of the items that assessed both dissociation and somatization scales served as indexes of dissociation and somatization symptoms severity, respectively. Cronbach alpha for the dissociation scale was .86 in the combined sample (Faroese =.89; Indian=.77) and Cronbach alpha for the somatization scale was .80 in the combined sample (Faroese =.86; Indian=.74) was good.

Analytic strategy

Data analysis was conducted using the IBM SPSS Statistics for Windows (version 29). Bi-variate relationships between the study variables were conducted through multiple Pearson correlation analyses. Coefficients ranging between ± 0.50 and ± 1 indicate a strong correlation;

coefficients ranging between ± 0.30 and ± 0.49 indicate a medium correlation; and, a coefficients below +. 29 indicate a a small correlation (Cohen, 1988). Considering differences between both samples on the level of exposure to ACE and subsequent impact on study variables, a *t*-test was conducted to compare the mean of direct and indirect exposure to ACE, defense mechanisms, dissociation and somatization symptoms between the Faroese and Indian adolescents with a Bonferroni correction.

To test our hypothesis of serial mediation, multiple step mediation methodology with a bootstrapped confidence interval for indirect effects was conducted (Model 6; Hayes, 2013). The examination of the models was performed through structural equation modeling (SEM). Specifically, the following was examined: (a) if both direct and indirect exposure to ACE were directly linked to somatization symptoms; (b) if direct and indirect exposure to ACEE were indirectly linked to somatization symptoms through mature, neurotic and immature defense mechanisms; (c) if direct and indirect exposure to ACE were indirectly linked to somatization symptoms through dissociation symptoms; and (d) if a two-step mediation process existed in which direct and indirect exposure to PTE was indirectly linked to somatization symptoms through defense mechanisms and dissociation.

Considering our hypothesis that the direct association between exposure to ACE and somatization symptoms would be different in both countries, a first model was tested in which sample membership (Faroese vs Indian sample) was a moderator in the test of direct path from both direct and indirect exposure to ACE to somatization symptoms. If sample membership moderates the relationship between the variables, the sample will be separated into Faroese and Indian samples, and the analysis of the models will be conducted separately for each group. There were no missing values in the tested variables.

Finally, to test the serial mediation model, a SEM (Hoyle & Smith, 1994) strategy utilizing AMOS software (Version 26; Arbuckle, 2012) and the Maximum Likelihood method was employed. The following criteria for SEM models fit were adopted: (a) $\chi 2$ test should be non-significant; (b) comparative fit index (CFI), normed fit index (NFI), (e) Tucker Lewis Index (TLI) > 0.95; (c) root mean square error of approximation (RMSEA) and standardized root mean square residual (SRMR) <0.08. A bootstrapped confidence interval of 5,000 bootstrapped

samples for the indirect effect was adopted. Analyses only included participants who had undergone at least one traumatic event. To assess significance of indirect paths, a bootstrapped confidence interval for the ab indirect effect, employing Preacher and Hayes' (2008) procedures, was adopted. A total of 5,000 bootstrapped samples was obtained to estimate indirect effects of each mediator. We computed bias corrected, accelerated 95% confidence intervals (CIs) to measure statistical significance for each mediator's "ab" paths and the two-step mediation. A confidence interval that does not include zero reflects evidence of a significant indirect effect or significant mediation.

Results

Prevalence of exposure to traumatic events

In the full sample, the most reported event was indirect exposure to a traffic accident, followed by direct and indirect exposure to the death of someone close, and indirect exposure to serious illness. Least prevalent was indirect exposure to other events, followed by direct exposure to rape and pregnancy /abortion. In the Faroese sample, the most common event was direct exposure to the death of someone close, followed by indirect exposure to traffic accidents and death of someone close. Least prevent reported event was direct exposure to pregnancy /abortion, followed by indirect exposure to other events, and direct exposure to rape, childhood neglect and sexual abuse. In the Indian sample, indirect exposure to a traffic accident was the most reported event, followed by direct and indirect exposure to the death of someone close. Least reported event was indirect exposure to other events, followed by direct exposure to rape, attempted suicide and divorce (Table 2).

Table 2

Potential trauma events and life events according to exposure and country membership

	Direct exposure in percentage			Indirect exposure in percentage		
	Faroese	Indian	Full sample	Faroese	Indian	Full sample

Event						
Traffic accident	16.7	39.2	25.1	50.2	52.1	51.1
Other serious accidents	11.6	17.0	13.7	30.4	32.1	31.1
Physical assault	9.6	7.8	8.9	26.3	10.7	20.5
Rape	4.1	1.2	3.0	14.3	3.6	10.3
Witnessed other people injured or killed	9.6	18.5	12.9	14.3	21.4	16.9
Came close to being injured or killed	12.7	18.2	14.8	11.9	15.1	13.1
Threats of violence	31.6	10.5	23.7	27.5	12.2	21.8
Near-drowning	21.8	8.5	16.8	15.6	8.3	12.8
Attempted suicide	9.9	2.4	7.1	21.5	10.2	17.3
Robbery/theft	13.7	10.9	12.7	27.5	24.6	26.4
Pregnancy /abortion	3.1	3.4	3.2	15.9	19.2	17.1
Serious illness	12.8	27.5	18.3	39.4	37.5	38.7
Death of someone close	52.7	41.4	48.5	46.6	43.1	45.3
Divorce	13.1	2.4	9.1	32.9	6.8	23.1
Sexual abuse	5.1	2.7	4.2	13.2	3.9	9.7
Physical abuse	7.3	6.1	6.8	15.9	7.5	12.8
Childhood neglect	4.9	4.4	4.7	16.6	10.0	14.1

Bullying	30.1	11.7	23.2	34.8	11.7	26.1
Absence of a parent	14.7	8.3	13.2	20.5	13.1	17.8
Other events	7.0	4.6	6.1	3.6	0.7	2.6

Eighty-nine participants (8.1%) did not report at least one adverse experience, of which 66 were Faroese adolescents (40 males and 26 females) and 23 were Indian adolescents (10 males and 13 females). These participants were excluded from subsequent analyzes. The average number of reported events in the total sample per participant was 7.1 (SD = 5.8; range 0-37). The average number of direct exposure was 2.8 (SD = 2.9; range 0-20) and the average number of indirect exposure was 4.3 (SD = 4.0; range 0-20). A t-test was conducted to compare the mean of exposure to traumatic events between the two samples. All data were tested for normality prior to the analyses using Kolmogorov-Smirnov test, as well as Levene's test for the homogeneity of the variance, and both assumptions were met. There were statistically significant differences between both samples in the total number of exposure to traumatic events ($t(1096) = 4.96$; $p < .001$), in the number of direct events ($t(1096) = 2.54$; $p < .05$), and in the number of indirect events ($t(1096) = 5.34$; $p < .001$). The mean of total exposure to traumatic events was higher in Faroese (M = 7.7; SD = 6.3; 95% IC: 7.2–8.2) compared to Indian (M = 5.9; SD = 4.8; 95% IC: 5.5–6.4); the mean of direct exposure to traumatic events was higher in Faroese (M = 2.9; SD = 3.1; 95% IC: 2.7–3.2) compared to Indian (M = 2.5; SD = 2.5; 95% IC: 2.2–2.7); the mean of indirect exposure to traumatic events was higher in Faroese (M = 4.8; SD = 4.3; 95% IC: 4.5–5.1) compared to Indian (M = 3.5; SD = 3.3; 95% IC: 3.1–3.8).

Intercorrelations between study variables

As can be seen in Table 3, the direct exposure to ACE presented a medium to positive correlation with indirect exposure to ACE and dissociation symptoms, and it was weakly positively linked to immature defense mechanisms and somatization symptoms. Indirect exposure to ACE was weakly positively associated with neurotic and immature defense mechanisms, and dissociation and somatization symptoms. Mature defense mechanisms had a medium positive association with neurotic and immature defense mechanisms, and it was weakly

negatively linked to somatization symptoms. Neurotic defense mechanisms presented a medium positive association with immature defense mechanisms, and it was weakly positively linked to dissociation and somatization symptoms. Immature defense mechanisms has a medium positive association with dissociation symptoms and it was weakly positively linked to somatization symptoms. Dissociation symptoms had a strong positive association with somatization symptoms. The remaining associations were non-significant,

Table 3

Correlation Matrix of Study Variables

Variable	1	2	3	4	5	6	7
1. Direct exposure to ACE	-	.34***	-.06	.01	.22***	.33***	.28***
2. Indirect exposure to ACE		-	.03	.07*	.19***	.24***	.22***
3. Mature defense mechanisms			-	.46***	.39***	.01	-.10**
4. Neurotic defense mechanisms				-	.37***	.14***	.07*
5. Immature defense mechanisms					-	.40***	.26***

6. Dissociation symptoms	-	.63***
7.Somatization symptoms		-

Note. ACE = Adverse childhood experiences. *$p < .05$. **$p < .01$. ***$p < .001$.

Mean comparisons between Faroese and Indian samples

As can be seen in Table 4, there were no statistically significant differences between Faroese and Indian participants on immature defense mechanisms. There were statistically significant differences between both samples on the remaining variables. The mean of mature and neurotic defense mechanisms was higher in Indian participants compared to Faroese participants; the mean of dissociation and somatization symptoms was higher in Faroese participants compared to Indian participants.

Table 4

Means, Standard Deviations and Mean Differences between Faroese and Indian sample on defense styles, dissociation symptoms, and somatization symptoms

	Faroese (*n*=621)		Indian (*n*=388)			
Variable	*M*	*SD*	*M*	*SD*	*t*	*d*
Mature style	39.17	10.97	46.26	9.40	-10.54***	.68
Neurotic style	42.20	11.07	45.67	9.82	-5.06***	.33
Immature style	99.44	26.05	102.46	20.96	-1.93	.13
Dissociation symptoms	12.40	3.73	11.30	3.31	4.74***	.31

Somatization symptoms	7.10	2.53	5.84	2.01	8.26***	.53

***$p < .001$.

Analysis of serial mediation

Considering our hypothesis that country membership was a moderator of the link between exposure to ACE and somatization symptoms, a model was specified in which direct and indirect exposure to ACE to somatization symptoms clusters was moderated by country membership (Faroese vs Indian sample). This model fit the observed data well (χ^2 (1) =.77, p = .38; NFI= 1.0; CFI =1.0; TLI = 1.0; RMSEA =0.0; SMSR =.01. The direct paths from country membership to direct exposure to ACE (b = -.62, p <.01, 95% CI, -.98, --.26) and indirect exposure to ACE (b = -1.63, p <.01, 95% CI, -2.12, -1.14) were significant. For this reason, subsequent analyses were conducted separately for Faroese and Indian samples.

Faroese sample

The existence of significant direct relations between direct and indirect exposure to ACE and somatization symptoms was confirmed first. This model fit the observed data well (χ^2 (1) =3.65, p = .06; NFI= .97; CFI =.98; TLI = .95; RMSEA =.06; SMSR =.03). The direct exposure to ACE (b = .19, p <.01, 95% CI, .13, .25) and the indirect exposure to ACE (b = .08, p <.05, 95% CI, .03, .13) were associated significantly with higher levels of somatization symptoms.

Next, a model was specified in which direct and indirect exposure to ACE had direct paths to somatization symptoms, as well as indirect paths through defense mechanisms (one-step mediation). The mediation model fit the observed data satisfactorily (χ^2 (1) =1.05, p = .31; NFI=. 1.0; CFI =1.0; TLI = 1.0; RMSEA =.01; SMSR =.01). The direct paths from direct exposure to ACE to somatization symptoms (b = .15, p <.01, 95% CI, .08, .22) remained significant when defense mechanisms were included in the model, but the direct paths from indirect exposure to ACE to somatization symptoms (b = .03, p =.31, 95% CI, -.02, .08) were no longer significant. When applying the bootstrapping analysis, total indirect effect from direct exposure to ACE (b = 1.37, p <.01, 95% CI, .69, 2.05) and indirect exposure to ACE (b = 1.08, p <.01, 95% CI, .59,

1.57) through immature defense mechanisms to somatization symptoms were significant. However, higher levels of mature defense mechanisms were associated with lower levels of somatization symptoms (*b* =-.17, *p* <.01, 95% CI, -.13, .21). The remaining paths were non-significant.

Following, it was examined if direct and indirect exposure to ACE had direct paths to somatization symptoms, as well as indirect paths through the dissociation variable. The mediation model fit the observed data well (χ^2 (2) = 5.82, *p* = .06; NFI= .99; CFI =.99; TLI = .97; RMSEA =.05; SMSR =.02). The direct paths from direct exposure to ACE somatization symptoms (*b* = .08, *p* <.05, 95% CI, .02, .14) remained significant when the model included dissociation symptoms, and the direct paths from indirect exposure to ACE to somatization symptoms (*b* = -.01, *p* =.94, 95% CI, -.05, .03) were non-significant. When applying the bootstrapping analysis, total indirect effect from direct exposure to ACE (*b* = .32, *p* <.01, 95% CI, .22, .42) and indirect exposure to ACE (*b* = .14, *p* <.01, 95% CI, .07, .21) through dissociation to somatization symptoms were significant. Thus, direct and indirect exposure to ACE were significantly associated with higher levels of dissociation symptoms, which were then associated with higher levels of somatization symptoms.

Finally, a model was specified in which direct and exposure to ACE had direct paths to somatization symptoms; one-step indirect paths through the defense mechanisms; and two-step indirect paths through defense mechanisms and the dissociation variable. Unstandardized coefficients and bootstrap solutions are presented in Table 5, and unstandardized results are presented in Figure 1. The observed data fit the mediational model well (χ^2 (1) = .01, *p* = .97; NFI=. 1.0; CFI =1.0; TLI = 1.0; RMSEA =0.0; SMSR =0.0). The direct paths from direct and indirect exposure to ACE to somatization symptoms were not significant when the model included all mediators. Specifically, the two-step indirect effects results indicated that direct and indirect exposure to ACE were significantly associated with higher levels of immature defense mechanisms, which were associated with higher levels of dissociation symptoms, which in turn were associated with higher levels of somatization symptoms. It was observed that only the indirect effect from indirect exposure to ACE to somatization symptoms through mature defense mechanisms was significant. Explicitly, higher levels of indirect exposure to ACE were associated with higher levels of mature defense mechanisms, which in turn were associated with

lower levels of somatization symptoms. It was also observed that the indirect effect from direct and indirect exposure to ACE to somatization symptoms through dissociation symptoms was significant. The results indicated that direct and indirect exposure to ACE were significantly associated with higher levels of dissociation symptoms, which in turn were associated with higher levels of somatization symptoms.

After omitting non-significant paths (i.e., direct exposure to ACE → somatization symptoms; indirect exposure to ACE → somatization symptoms; neurotic defense mechanisms → somatization symptoms; immature defense mechanisms → somatization symptoms; direct exposure to ACE → mature defense mechanisms; mature defense mechanisms → dissociation symptoms; neurotic defense mechanisms → dissociation symptoms) our final model fit the observed data well (χ^2 (7) = 10.31, p = .17; NFI= .99; CFI = 1.0; TLI = .99; RMSEA =.03; SMSR =.03).

Table 5

Bootstrapped point estimate for direct and indirect effects and 95% confidence intervals for predicting somatization symptoms by sums of direct and indirect exposure to traumatic events through defense styles and dissociation symptoms in Faroese sample

	Point estimate	SE	BCa 95% CI (lower, upper)
Somatization symptoms			
Direct effect of direct exposure	.06	.03	(.00, .12)
Direct effect of indirect exposure	.00	.02	(-.04, .04)
Indirect effect of direct exposure via MDM	.27	.15	(-.56, .02)
Indirect effect of indirect exposure via MDM	-.35	.02	(-.41, -.29)**

Indirect effect of direct exposure via NDM	.01	.01	(-.01, .03)
Indirect effect of indirect exposure via NDM	.13	.11	(-.09, .35)
Indirect effect of direct exposure via IDM	.07	.04	(-.01, .15)
Indirect effect of indirect exposure via IDM	.01	.01	(-.01, .03)
Indirect effect of direct exposure via dissociation symptoms	.24	.05	(.15, .33)***
Indirect effect of indirect exposure via dissociation symptoms	.07	.03	(.01, .13)*
Indirect effect of direct exposure via MDM and dissociation symptoms	-.03	..02	(-.06, .00)
Indirect effect of indirect exposure via MDM and dissociation symptoms	.08	.05	(-.18, .02)
Indirect effect of direct exposure via NDM and dissociation symptoms	.05	.04	(-.03, .13)
Indirect effect of indirect exposure via NDM and dissociation symptoms	.02	.02	(-.02, .02)
Indirect effect of direct exposure via IDM and dissociation symptoms	.40	.03	(.35, .45)***

Indirect effect of indirect exposure via IDM and dissociation symptoms	.37	.04	(.29, .45)***

Note. MDM = Mature Defense Mechanisms; NDM = Neurotic Defense Mechanisms; IDM = Immature Defense Mechanisms; BCa = bias corrected and accelerated; CI = confidence intervals; Confidence intervals that do not include 0 (null association) are significant. *p < .05. **p < .01. ***p < .001.

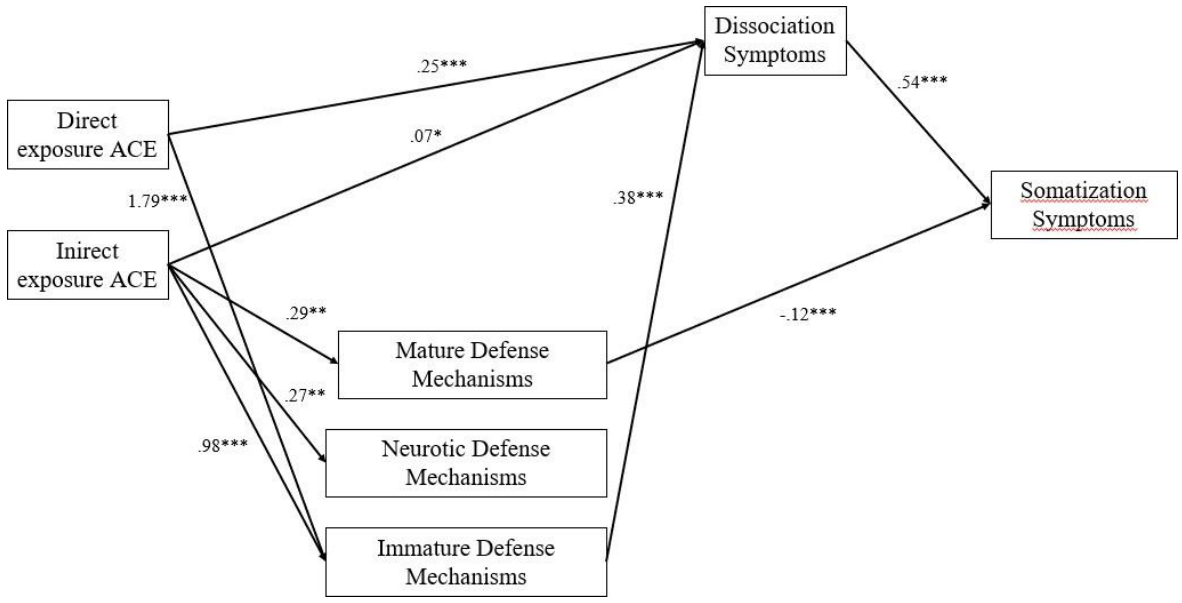

Figure 1. A Serial Mediational Integrated Model for Somatization Symptoms by Defense Mechanisms and Dissociation Symptoms in Faroese Sample. Rectangles indicate measured variables. Unidirectional arrows depict hypothesized directional links. Standardised maximum likelihood parameters are used. N =621; *p <.05, **p <.01, ***p <.001.

Indian sample

The model in which was tested the existence of significant direct paths between direct and indirect exposure to ACE and somatization symptoms fit the observed data well (χ^2 (1) = 2.93, *p* = .09; NFI= .98; CFI =.98; TLI = .95; RMSEA =.06; SMSR =.03). The direct exposure to ACE (*b* = .18, *p* <.01, 95% CI, .10, .26) and the indirect exposure to ACE (*b* = .09, *p* <.05, 95% CI, .03, .15) were associated significantly with higher levels of somatization symptoms.

Following, a model was tested in which direct and indirect exposure to ACE had direct paths to somatization symptoms, as well as indirect paths through defense mechanisms (one-step mediation). The mediation model fit the observed data satisfactorily (χ^2 (1) =.32, *p* = .57; NFI=. 1.0; CFI =1.0; TLI = 1.0; RMSEA =0.0; SMSR =.01). The direct paths from direct exposure to ACE to somatization symptoms (*b* = .10, *p* <.05, 95% CI, -.02, .18) and indirect exposure to ACE symptoms (*b* = .09, *p* <.05, 95% CI, .03, .15) to somatization symptoms remained significant when defense mechanisms were included in the model. When applying the bootstrapping analysis, total indirect effect from direct exposure to ACE through immature defense mechanisms (*b* = 2.33, *p* <.01, 95% CI, 1.39, 3.27) and neurotic defense mechanisms (*b* = .55, *p* <.05, 95% CI, .13, .97) to somatization symptoms were significant. The remaining paths were non-significant.

The model in which direct and indirect exposure to ACE had direct paths to somatization symptoms, as well as indirect paths through the dissociation variable fit the observed data well (χ^2 (2) = 3.84, *p* = .15; NFI= .99; CFI =.99; TLI = .98; RMSEA =.05; SMSR =.03). The direct paths from direct exposure to ACE somatization symptoms (*b* = .01, *p* =.90, 95% CI, -.06, .08) were non-significant, but the direct paths from indirect exposure to ACE somatization symptoms (*b* = .05, *p* <.05, 95% CI, .01, .09) remained significant when the model included dissociation symptoms. When applying the bootstrapping analysis, total indirect effect from direct exposure to ACE (*b* = .43, *p* <.01, 95% CI, .30, .56) and indirect exposure to ACE (*b* = .10, *p* <.05, 95% CI, .01, .19) through dissociation to somatization symptoms were significant. Thus, direct and indirect exposure to ACE were significantly associated with higher levels of dissociation symptoms, which were then associated with higher levels of somatization symptoms.

A final model was analyzed in which direct and exposure to ACE had direct paths to somatization symptoms; one-step indirect paths through the defense mechanisms; and two-step indirect paths through defense mechanisms and the dissociation variable. Unstandardized coefficients and bootstrap solutions are presented in Table 6, and unstandardized results are presented in Figure 2. The observed data fit the mediational model well (χ^2 (2) = .36, p = .84; NFI=. 1.0; CFI =1.0; TLI = 1.0; RMSEA =0.0; SMSR =.01). The direct paths from indirect exposure to ACE to somatization symptoms were remained significant when the model included all mediators, but the direct paths from direct exposure to ACE to somatization symptoms were not significant. The two-step indirect effects results indicated that direct exposure to ACE was significantly associated with higher levels of both neurotic and immature defense mechanisms, which were associated with higher levels of dissociation symptoms, which in turn were associated with higher levels of somatization symptoms. It was also observed that indirect exposure to ACE was significantly associated with higher levels of immature defense mechanisms, which were associated with higher levels of dissociation symptoms, which in turn were associated with higher levels of somatization symptoms. It was also observed that the indirect effect from direct and indirect exposure to ACE to somatization symptoms through dissociation symptoms was significant. The results indicated that direct and indirect exposure to ACE were significantly associated with higher levels of dissociation symptoms, which in turn were associated with higher levels of somatization symptoms.

After omitting non-significant paths (i.e., direct exposure to ACE → somatization symptoms; mature defense mechanisms → somatization symptoms; neurotic defense mechanisms → somatization symptoms; immature defense mechanisms → somatization symptoms; direct exposure to ACE → mature defense mechanisms; indirect exposure to ACE → mature defense mechanisms; indirect exposure to ACE → neurotic defense mechanisms) our final model fit the observed data well (χ^2 (8) = 7.47, p = .49; NFI= .99; CFI = 1.0; TLI = 1.0; RMSEA =0.0; SMSR =.02)

Table 6

Bootstrapped point estimate for direct and indirect effects and 95% confidence intervals for predicting somatization symptoms by sums of direct and indirect exposure to traumatic events through defense styles and dissociation symptoms in Indian sample

	Point estimate	SE	BCa 95% CI (lower, upper)
Somatization symptoms			
Direct effect of direct exposure	-.01	.03	(-.08, .06)
Direct effect of indirect exposure	.05	.02	(.01, .09)*
Indirect effect of direct exposure via MDM	-.12	.21	(-.53, .29)
Indirect effect of indirect exposure via MDM	.23	.15	(-.07, .53)
Indirect effect of direct exposure via NDM	.01	.01	(-.01, .03)
Indirect effect of indirect exposure via NDM	.14	.16	(-.17, .45)
Indirect effect of direct exposure via IDM	.01	.02	(-.03, .05)
Indirect effect of indirect exposure via IDM	.36	.39	(-.40, 1.12)
Indirect effect of direct exposure via dissociation symptoms	.29	.06	(.16, .42)***

Indirect effect of indirect exposure via dissociation symptoms	.09	.05	(.01, .17)*
Indirect effect of direct exposure via MDM and dissociation symptoms	-.03	.07	(-.11, .17)
Indirect effect of indirect exposure via MDM and dissociation symptoms	.01	.05	(-.09, .11)
Indirect effect of direct exposure via NDM and dissociation symptoms	.55	.06	(.13, .97)**
Indirect effect of indirect exposure via NDM and dissociation symptoms	.06	.08	(-.09, .21)
Indirect effect of direct exposure via IDM and dissociation symptoms	.25	.09	(.09, .41)***
Indirect effect of indirect exposure via IDM and dissociation symptoms	.08	.03	(.03, .13)*

Note. MDM = Mature Defense Mechanisms; NDM = Neurotic Defense Mechanisms; IDM = Immature Defense Mechanisms; BCa = bias corrected and accelerated; CI = confidence intervals; Confidence intervals that do not include 0 (null association) are significant. *$p < .05$. **$p < .01$. ***$p < .001$.

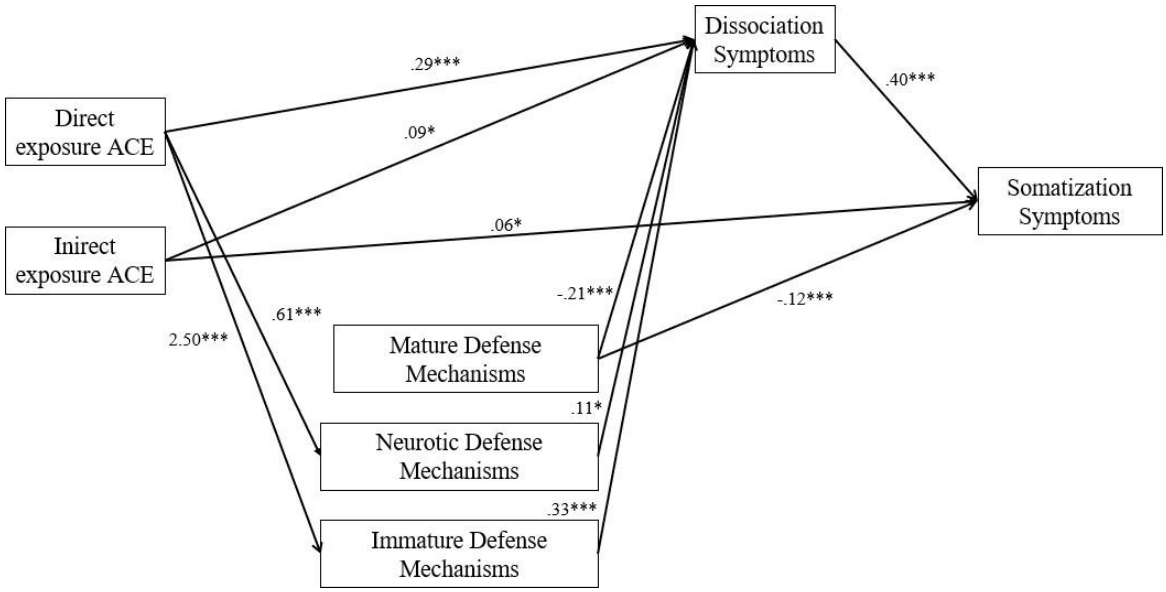

Figure 2. A Serial Mediational Integrated Model for Somatization Symptoms by Defense Mechanisms and Dissociation Symptoms in Indian Sample. Rectangles indicate measured variables. Unidirectional arrows depict hypothesized directional links. Standardised maximum likelihood parameters are used. N =388; *p <.05, **p <.01, ***p <.001.

Discussion

The main goal of the current study was to examine the effect of direct and indirect exposure to ACE on somatization symptoms through the mediation of defense mechanisms and dissociation in Faroese and Indian adolescents. The current findings indicate that the association between these variables were different for both samples. Our results demonstrated that dissociation symptoms were mediators of the association between direct and indirect exposure to ACE and somatization symptoms in both samples. Regarding the other variables under study, the differences observed between the samples will be described next. In the Faroese sample, only mature defense mechanisms were mediators of the association between indirect exposure to ACE and somatization symptoms. Additionally, serial multiple mediation model results indicated that higher direct and indirect exposure to ACE were significantly associated with higher levels of immature defense mechanisms, which were associated with higher levels of dissociation

symptoms, which in turn were associated with higher levels of somatization symptoms. In the Indian sample, defense mechanisms were not mediators of the association between direct and indirect exposure to ACE and somatization symptoms. For this sample, serial multiple mediation model results indicated that direct exposure to ACE was significantly associated with higher levels of immature defense mechanisms, which were associated with higher levels of dissociation symptoms, which in turn were associated with higher levels of somatization symptoms. To the best of our knowledge, this is the first study to analyze the joint mediating role of defense mechanisms and dissociation in adaptation to multiple adverse experiences in Faroese and Indian adolescents. Our hypotheses will be discussed in turn.

Our first hypothesis was that country membership would moderate the link between exposure to ACE and somatization symptoms. This hypothesis was fully supported. The results of the moderation analysis showed that the relationship between exposure to ACE and somatization symptoms was dependent on country membership, which was to be expected due to the differences in the context of the Faroe Islands and India. Further analyses were performed separately, as the moderating effect was confirmed and there were significant differences between both samples on the variables under study, which will be discussed next.

The following might provide a potential rationale for the observed moderation effect. The current study indicated that Faroese participants presented higher direct and indirect exposure to ACE, compared to Indian participants. These results were surprising, since previous studies have indicated that youth living in LALMIC have a higher prevalence of exposure to multiple forms of adversity compared to those living in high-income countries (Le et al., 2018). The Indian context also includes extreme poverty, collective violence and natural disasters, thus, exposure to adverse experiences was expected to be higher (Rasmussen et al., 2013). A possible explanation for these findings could be that Indian participants predominantly came from middle- and upper-class backgrounds, and could have been brought up in a more protected environment. Additionally, cultural factors might contribute to these results, including the acceptance of certain events within Indian society, potentially leading to underreporting of traumatic experiences. For instance, corporal punishment remains prevalent as a disciplinary method employed by parents and schools, and is therefore not regarded as a violent act (Deb et al., 2016b; Rasmussen et al., 2013).

Moreover, the higher prevalence of exposure to ACE among Faroese adolescents may be attributed to the cultural context in the country. For instance, in Faroese culture, adolescents are often encouraged by their families to be independent, self-reliant and to seek support from peers (Gaini, 2013). However, this emphasis on independence, paired with a lack of parental guidance and supervision (Gaini, 2013), can potentially make Faroese adolescents more vulnerable to being exposed to ACE. Conversely, in India, members of the family are invested in one another and are closely knit together. The family also plays a crucial role in protecting, nurturing, and supporting children (Sooryamoorthy, 2012). This familial support and involvement can serve as a protective factor against the exposure to ACE among Indian adolescents. Nonetheless, even though Faroese participants reported more ACE, these were less violent in nature. Meanwhile, Indian participants reported more violent events, including serious accidents and witnessing other people injured or killed. Despite coming from a privileged background, it is plausible that Indian adolescents from this sample are still exposed to the adversity-prone environment that characterizes India (Saraswathi & Oke, 2013). This also further explains the moderation effect of country membership confirmed in the present study.

There were also significant differences between both samples on somatization symptoms. Faroese participants presented more somatization symptoms when compared to Indian participants, which may be explained by the following hypothesis. First, lower somatization symptoms among the Indian participants may be attributed to the specific events reported by this sample. Given the more violent and life-threatening nature of the events, it is reasonable to suggest that they are more likely associated with the development of PTSD rather than somatization symptoms (Trickey et al., 2012). Secondly, higher levels of somatization in the Faroese sample can possibly be accounted for by lower levels of mature defense mechanisms and higher levels of dissociation symptoms among these participants. This goes in accordance with studies on the association between dissociation and somatization symptoms that indicate that individuals who experience dissociation in response to traumatic events are prone to manifest their psychological distress through physical symptoms (Farley & Keaney, 1997; Luoni et al., 2018; Salmon et al., 2003). Similarly, studies on the association between defense mechanisms and somatization symptoms, also indicate that lower levels of mature defense mechanisms are associated with higher levels of somatization symptoms (Hyphantis et al., 2013a; Hyphantis et al., 2013b; Waikamp et al., 2021).

Furthermore, differences on the levels of defense mechanisms and dissociation symptoms between the samples can be explained as follows. On the one hand, higher levels of exposure to traumatic events on the Faroese sample can lead to a perception of reduced control over their experiences, which may result in a regression of mature defense mechanisms to more primitive or less adaptive ones (Punamäki et al., 2002). This may explain lower levels of mature defense mechanisms on this sample. On the other hand, existing research has consistently shown that interpersonal potentially traumatic events such as sexual trauma, having been threatened or physically abused and bullied are typically associated with more dissociation symptoms (Gušić et al., 2016; Chu & Dill, 1990). Importantly, these types of events are more prevalent among the Faroese sample, which provides a plausible explanation for higher levels of dissociation symptoms in these participants. In turn, these differences can affect the relationship between exposure to ACE and somatization symptoms, which will be discussed below.

Our second hypothesis was that higher direct and indirect exposure to ACE would be associated with higher levels of somatization symptoms. This hypothesis was partially supported. In the Faroese sample, results indicated that there was a significant association between these variables, that is, higher direct and indirect exposure to ACE were associated significantly with higher levels of somatization symptoms. However, when testing for direct paths, the direct paths from direct and indirect exposure to ACE to somatization symptoms were no longer significant when the mediators were included in the model. In the Indian sample, results also indicated that higher direct and indirect exposure to ACE were associated significantly with higher levels of somatization symptoms, but only direct paths from indirect exposure to ACE to somatization symptoms remained significant when the mediators were included in the model. Findings from both samples support prior studies which have proposed that other psychological variables mediate the association between multiple exposure to adverse experiences and somatization symptoms (Guerra et al., 2016; Salmon et al., 2003).

In addition, results also indicate that indirect exposure to ACE is directly associated with somatization symptoms in Indian participants. Previous studies have mainly analyzed the association between direct exposure to adverse events and somatization. Conversely, indirect exposure to traumatic events has commonly been associated with PTSD. Our findings indicate that it can have other effects on mental health, specifically, results revealed that multiple indirect

exposure to ACE is a risk factor for the development of somatization symptoms. It can be proposed that multiple indirect exposure to ACE can cause a constant state of threat, ambiguity and uncertainty regarding one's faith (Petersen et al., 2010), which can lead to chronic stress responses (Fischer et al., 2014). In order to regulate the body's response to stress, the stress system, which is made up of multiple interconnected mind-body systems, such as the hypothalamic-pituitary-adrenal (HPA) axis, is activated. Chronic stress can disrupt the normal functioning of these systems, leading to dysregulation, which can have various effects on the body, including impairments in immune function, inflammation, and alterations in neurotransmitter systems. These physiological changes can contribute to the development or exacerbation of somatic symptoms (Kozlowska et al., 2013). These results highlight the importance of differentiating the exposure criteria used in the present study. Furthermore, future studies should further analyze the association between indirect exposure to ACE and somatization symptoms.

Our third hypothesis was that defense mechanisms would mediate the link between direct and indirect exposure to ACE and somatization symptoms. This hypothesis was partially supported. To begin with, our findings reveal that immature defense mechanisms did not mediate the link between direct and indirect exposure to ACE and somatization symptoms in both samples. These results suggest that in the context of multiple exposures to ACE, the role of immature defense mechanisms in adolescents might be to aggravate dissociation symptoms, which then increase somatization symptoms. Immature defense mechanisms may hinder the ability to adjust to the distressing and overwhelming nature of ACE. As a result, adolescents may be more likely to rely on dissociation as a way to mentally escape or disconnect from distressing thoughts, feelings, or memories associated with the ACE. Consequently, dissociated traumatic memories can later be expressed in somatic symptoms (Powers et al., 2015; Sandstorm & Cramer, 2003; Saxe et al., 1994).

Moreover, mature defense mechanisms mediated the association between indirect exposure to ACE and somatization symptoms in the Faroese sample. Specifically, results indicate that higher levels of indirect exposure to ACE were associated with higher levels of mature defense mechanisms, which were then associated with lower levels of somatization symptoms. One plausible explanation for these findings is that indirect exposure to ACE, which

does not directly pose a threat to one's life, may foster mentalization. Mentalization refers to the capacity to accurately perceive and comprehend one's own and others' mental states, as well as distinguish between internal and external realities without distortions (Tanzilli et al., 2021). In turn, enhanced mentalizing abilities are known to enable individuals to employ adaptive strategies, such as mature defense mechanisms, to regulate their emotional states and cope with distressing experiences (Fonagy et al., 2018). Consequently, previous research has found that a predominant use of adaptive defenses is associated with lower levels of somatic symptoms (Hyphantis et al., 2013a; Hyphantis et al., 2013b; Waikamp et al., 2021).

Furthermore, in the Indian sample, mature defense mechanisms did not mediate the association between direct and indirect exposure to ACEs and somatization symptoms. It is noteworthy that previous studies that reported mediation of defense mechanisms were from samples of adults (Finzi-Dottan & Karu, 2006; Nickel and Egle, 2006). Additionally, the current study is the first to test defense mechanisms as a mediator between ACE and somatization symptoms among Faroese and Indian adolescents.

Our fourth hypothesis was that dissociation symptoms would mediate the association between direct and indirect exposure to ACEs and somatization symptoms. This hypothesis was fully supported for both samples. Indirect effect of direct and indirect exposure to ACEs on somatization symptoms through dissociation symptoms was significant. These findings demonstrate that higher direct and indirect exposure to ACE were associated with higher levels of dissociation symptoms, which were then associated with higher levels of somatization symptoms. Our findings confirm previous research on the relationship between these variables (Chu & Dill, 1990; Salmon et. al, 2003; Saxe et al., 1994) which has indicated that memories of the traumatic experiences are dissociated from consciousness as a coping response to deal with the exposure to these painful events, but later manifest in somatic symptoms.

Our fifth and final hypothesis was that serial multiple mediation models would indicate that direct and indirect exposure to ACE were associated with higher levels of immature defense mechanisms and lower levels of mature defense mechanisms, that were relate to higher levels of dissociation symptoms, which were then related to higher levels of somatization symptoms. This hypothesis was partially supported.

In the Faroese sample, results indicated that direct and indirect exposure to ACE were related to higher levels of immature defense mechanisms, which were associated with higher levels of dissociation symptoms, which in turn were associated with higher levels of somatization symptoms. These findings suggest that both direct and indirect exposure to ACE can lead to a regression of defense mechanisms, characterized by a reliance on immature defense mechanisms, which then result in higher dissociation and, consequently, higher somatization symptoms. By relying on immature defense mechanisms, individuals may face difficulties in processing and integrating their experiences, leading to their dissociation from consciousness. Consequently, trauma-related memories are stored in an implicit level and have no verbal component, which interferes with the disclosure of the traumatic experience. Since they cannot be symbolized, individuals can still experience bodily sensations associated with these memories. Thus, somatic symptoms can arise or aggravate, as an unconscious representation of the traumatic experiences (Saxe et al., 1994; van der Kolk & Fisler, 1995).

In the Indian sample, results indicated that only direct exposure to ACE was related with higher levels of immature and neurotic defense mechanisms, which were associated with higher levels of dissociation and somatization. It seems that among Indian participants, the particular repetitive nature of direct exposure to ACE results in a process of regression to more primitive defenses, leading to higher dissociation and somatization symptoms. It is possible to propose that indirect exposure to ACE is viewed with normalcy in the Indian context, unlike the Faroese context, explaining the lack of a significant effect on the other variables under study. In addition, while mature defense mechanisms were not mediators, they were negatively associated with dissociation and somatization symptoms. These results suggest that mature defense mechanisms,which allow more conscious awareness of stressors, are protective mechanisms in the face of traumatic experiences (Perry et al., 2015; Vaillant, 2011) for this sample.

Our study is not exempt from limitations. First, the cross-sectional design does not allow us to infer causality. It is not possible to obtain causal inferences from the serial mediation analyses. In future studies, it would be beneficial to analyze the association between the variables using a longitudinal design. Second, the current study cannot make general conclusions about Indian youth as the sample used is not representative. Specifically, the participants were predominantly from middle- and upper-class socioeconomic backgrounds. Future studies should

collect data on adolescents from other socioeconomic levels. Third, the study relies on the use of self-reporting measures. Fourth, ACE report was retrospective, which may be subject to memory bias. Fifth, due to the study design it was not possible to identify whether an event occurred more than once, hence a distinction between the effects of a single traumatic event or repeated traumatic events could not be made. Sixth, the traumatic life-event questionnaire has not been validated and was initially created to reflect a European sample. Therefore, questions depicting a more complex context, where for example natural disasters and terrorism take place, might have been relevant in this study. It is also important to note that cultural differences in the interpretation of the psychological constructs being assessed may have affected the responses of the participants. Seventh, the TSC was primarily used to measure somatization symptoms. Future research should use measures based on DSM-5 criteria. Eighth, the data used in this study was collected in 2012. Therefore, there are possibly differences in the current context of both countries. Considering the limitations identified, our results need to be interpreted with caution and their generalizability should be tested by means of prospective studies and in different cultures as well. It is also recommended that future studies replicate this mediation hypothesis among adolescents from different cultures.

The current results suggest that exposure to ACE makes adolescence a vulnerable period of development for many Faroese and Indian youth. Our study suggests that polytraumatization increases the risk of mental health problems among adolescents, namely, somatization symptoms. Given that many individuals with somatic complaints seek primary healthcare, it is advisable for healthcare providers to assess trauma history, including both direct and indirect ACE exposure, and consider referral to mental health services when necessary. Furthermore, our results indicate the key role of both defense mechanisms and dissociation symptoms as underlying psychological mechanisms in the association between polytraumatization and somatization symptoms. These findings suggest that mental health professionals should pay attention to the use of defense mechanisms, especially immature defense mechanisms, and dissociation symptoms among adolescents following multiple exposure to ACE, since these clinical manifestations seem to increase the risk to develop somatic symptoms. Therefore, it is recommended that clinicians focus on addressing defenses that restrict awareness of thoughts, emotions, and behaviors, as well as the disruption and/or discontinuity of traumatic experiences

in adolescents exposed to adversity. Moreover, the identification of specific and potentially modifiable psychological processes may improve patient care and reduce healthcare costs.

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Overall Discussion

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Objectives and Hypotheses

The main goal of the current study was to analyze the effect of direct and indirect exposure to ACE on somatization symptoms through the mediation of defense mechanisms and dissociation in Faroese and Indian adolescents. The empirical article presented in this dissertation allowed us to successfully address the main objectives of our investigation: to analyze the association between direct and indirect exposure to ACE and somatization symptoms; to assess the mediating role of both defense mechanisms and dissociation symptoms in the relationship between direct and indirect exposure to multiple ACE; and to examine the series of associations between exposure to multiple ACE, defense mechanisms, dissociation symptoms and somatization symptoms. As presented in the results of our empirical study, the majority of our hypotheses were supported. In line with previous studies, our findings emphasized the role of defense mechanisms and dissociation as mediators of the association between exposure to ACE and somatization (Finzi-Dottan & Karu, 2006; Nickel & Egle, 2006; Salmon et al., 2003). Our findings indicate that the association between these variables were different for the Faroese and the Indian samples. First, results showed that dissociation symptoms were mediators of the association between direct and indirect exposure to ACE and somatization symptoms in both samples. Considering the other variables under study, it is important to note observed differences between both samples. In the Faroese sample, only mature defense mechanisms were mediators of the association between indirect exposure to ACE and somatization symptoms. In addition, serial multiple mediation model results indicated that higher direct and indirect exposure to ACE were significantly associated with higher levels of immature defense mechanisms, which were associated with higher levels of dissociation symptoms, which in turn were associated with higher levels of somatization symptoms. In the Indian sample, defense mechanisms were not mediators of the association between direct and indirect exposure to ACE and somatization symptoms. In turn, results from the serial multiple mediation model indicated that direct exposure to ACE was significantly associated with higher levels of immature defense mechanisms, which were associated with higher levels of dissociation symptoms, which in turn were associated with higher levels of somatization symptoms. To the best of our knowledge, this is the first study to analyze the joint mediating role

of defense mechanisms and dissociation in adaptation to multiple ACE in Faroese and Indian adolescents. Our hypotheses will be discussed next.

Our first hypotheses suggested that country membership would be a moderator in the association between direct and indirect exposure to ACE and somatization symptoms. The results presented in the empirical article of this dissertation indicated that this hypothesis was fully supported. The results of the moderation analysis demonstrated that the link between ACE exposure and somatization symptoms varied based on the participants' country of origin, as anticipated due to the distinct contexts of the Faroe Islands and India (Gaini, 2013, Gaini & Sleire, 2023; Rasmussen et al., 2013). Consequently, additional analyses were conducted separately, as the moderating effect was confirmed, and significant differences between both samples were observed concerning the variables under study, which will be elaborated upon in the following discussion.

Our findings revealed that Faroese participants presented higher levels of both direct and indirect exposure to ACE compared to Indian participants. These results are inconsistent with previous research that has indicated that youth living in LALMIC have a higher prevalence of exposure to multiple forms of adversity compared to those living in high-income countries (Le et al., 2018). The Indian context, characterized by extreme poverty, collective violence, and natural disasters, had led us to expect higher exposure to adverse experiences among the Indian participants (Rasmussen et al., 2013). One potential explanation for these unexpected findings could be that Indian participants predominantly came from middle- and upper-class backgrounds, providing a more protected upbringing. Furthermore, cultural factors might have contributed to the results, such as the acceptance of certain events within Indian society, potentially leading to underreporting of traumatic experiences. For example, corporal punishment has remained as a prevalent form of discipline applied by parents and schools, and it may not be regarded as a violent act within the cultural context (Deb et al., 2016b; Rasmussen et al., 2013). These findings highlight the importance of considering cultural and socioeconomic factors in understanding the variation in exposure to ACE across different samples, and further explain the observed moderation effect.

Furthermore, the higher prevalence of exposure to ACE among Faroese adolescents may be influenced by the cultural context of the Faroe Islands. In the Faroese culture, parenting often imposes autonomy and self-reliance on youth. Coupled with limited supervision and parental guidance, which is also common during adolescent years in the country's culture, Faroese adolescents may be more vulnerable to exposure to ACE (Gaini, 2013). On the other hand, in India, families are closely knit, and members are deeply invested in each other's well-being. The family unit plays a pivotal role in protecting, nurturing, and supporting children (Sooryamoorthy, 2012), which can serve as a protective factor against ACE exposure among Indian adolescents. It is important to note that even though Faroese participants reported a higher prevalence of ACE, these experiences were less violent in nature. In contrast, Indian participants reported more violent events, such as serious accidents and witnessing injuries or fatalities of others. This discrepancy could be attributed to the adversity-prone environment that characterizes India, which may still affect Indian adolescents, despite their privileged background (Saraswathi & Oke, 2013). These insights also support and explain the moderation effect of country membership as confirmed in the present study.

Significant differences were also observed between the two samples regarding somatization symptoms. Faroese participants exhibited higher levels of somatization symptoms in comparison to Indian participants, and these differences may be attributed to the following hypotheses. Firstly, lower somatization symptoms reported by Indian participants could be attributed to the specific events experienced by this sample. Given the more violent and life-threatening nature of the events reported, it is reasonable to suggest that they may be more closely associated with the development of PTSD rather than somatization symptoms (Trickey et al., 2012). Secondly, higher levels of somatization observed in the Faroese sample could be attributed to lower levels of mature defense mechanisms and higher levels of dissociation symptoms exhibited by these participants. These findings are in line with previous research on the association between defense mechanisms and somatization symptoms, which have indicated that lower levels of mature defense mechanisms are associated with higher levels of somatization symptoms (Hyphantis et al., 2013a; Hyphantis et al., 2013b; Waikamp et al., 2021). Likewise, former studies on the association between dissociation and somatization symptoms have also suggested that individuals who experience dissociation in response to traumatic events are more

likely to express their psychological distress through physical symptoms (Farley & Keaney, 1997; Luoni et al., 2018; Salmon et al., 2003).

Moreover, we provide an explanation for the observed differences on the levels of defense mechanisms and dissociation symptoms between the samples. Among the Faroese participants, higher levels of exposure to traumatic events may lead to a perception of reduced control over their experiences, potentially resulting in a regression of mature defense mechanisms towards more primitive or less adaptive ones (Punamäki et al., 2002). This could account for the lower levels of mature defense mechanisms observed in this sample. Additionally, prior research has consistently demonstrated that interpersonal potentially traumatic events such as sexual trauma, threats of physical abuse or actual physical abuse, as well as bullying, are typically associated with increased dissociation symptoms (Gušić et al., 2016; Chu & Dill, 1990). Notably, these types of events are more prevalent among the Faroese sample, offering a plausible explanation for higher levels of dissociation symptoms observed in these participants. The differences described can, in turn, affect the relationship between exposure to ACE and somatization symptoms, which will be discussed below.

Our second hypothesis was that higher direct and indirect exposure to ACE would be associated with higher levels of somatization symptoms. The results presented in the empirical article of this dissertation indicated that this hypothesis was partially supported. In the Faroese sample, our results showed that higher direct and indirect exposure to ACE were associated significantly with higher levels of somatization symptoms. Despite the significant association between both variables, when indirect paths were tested, the direct paths from direct and indirect exposure to ACE to somatization symptoms were no longer significant when the mediators were included in the model. In the Indian sample, results also showed a significant and positive association between direct and indirect exposure to ACE and somatization symptoms, but only direct paths from indirect exposure to ACE to somatization symptoms remained significant when the mediators were included in the model. These findings support prior studies which have suggested that other psychological variables mediate the association between multiple exposure to ACE and somatization symptoms (Guerra et al., 2016; Salmon et al., 2003).

Furthermore, results also indicated that indirect exposure to ACE is directly associated with somatization symptoms in Indian participants. It is worth noting that previous studies have

predominantly analyzed the association between direct exposure to traumatic events and somatization symptoms, and that indirect exposure has been mainly associated with PTSD. Our findings indicate that indirect exposure to ACE can have varied effects on mental health, including being a risk factor for the development of somatization symptoms. It is likely that multiple indirect exposure to ACE can cause a constant state of threat, ambiguity, and uncertainty regarding one's faith (Petersen et al., 2010), which can lead to chronic stress responses (Fischer et al., 2014). Consequently, the stress system, which is made up of multiple interconnected mind-body systems, such as the hypothalamic-pituitary-adrenal (HPA) axis, is activated in an attempt to regulate the body's response to stress. However, chronic stress can cause hyperactivation of these systems, leading to dysregulation, which can lead to the manifestation of severe somatic sequelae, including impairments in immune function, inflammation, and alterations in neurotransmitter systems. Thus, these physiological changes can contribute to the development or exacerbation of somatic symptoms (Kozlowska et al., 2013). Our results highlight the importance of differentiating the exposure criteria, as demonstrated in our study. Previous research has primarily focused on examining the effects of direct exposure to ACE either independently or in combination with indirect exposure to ACE. However, our study reveals that this differentiation is critical, as it yields distinct effects for both types of ACE exposure on mental health. Furthermore, it is essential for future studies to conduct more in-depth analyses concerning the association between indirect exposure to ACE and somatization symptoms, along with other psychological symptoms. For instance, when testing a similar model as in our study, further research could take into account defense mechanisms and dissociation, specifically psychoform and somatoform dissociation. This will lead to a more comprehensive understanding of the intricate relationship between indirect exposure to ACE and mental health outcomes.

Our third hypothesis was that defense mechanisms would mediate the link between direct and indirect exposure to ACE and somatization symptoms. The results presented in the empirical article of this dissertation indicated that this hypothesis was partially supported. Our results indicate that immature defense mechanisms did not mediate the association between direct and indirect exposure to ACE and somatization symptoms in adolescents from both samples. It is possible that the role of immature defense mechanisms in adolescence is to intensify dissociation symptoms, which then relate to increased somatization symptoms. In this sense, immature

defenses may impede an individual's ability to cope with the distressing and overwhelming nature of ACE. This difficulty in coping may lead them to resort to dissociation as a way to mentally escape or disconnect from the distressing thoughts, feelings, or memories associated with ACE. As a consequence, dissociated traumatic memories can later be expressed in somatic symptoms (Powers et al., 2015; Sandstorm & Cramer, 2003; Saxe et al., 1994).

Nevertheless, mature defense mechanisms mediated the link between indirect exposure to ACE and somatization symptoms in the Faroese sample. Our findings indicate that higher levels of indirect exposure to ACE were associated with higher levels of mature defense mechanisms, which were then associated with lower levels of somatization symptoms. One possible explanation for these results might be that indirect exposure to ACE, which does not constitute a direct threat to one's life, may promote mentalization. Mentalization refers to the ability to accurately understand and perceive one's own and others' mental states and distinguish between internal and external realities without distortions (Tanzilli et al., 2021). Mentalizing enables individuals to use adaptive strategies, like mature defense mechanisms, to regulate emotions and cope with distressing experiences, such as ACE (Fonagy et al., 2018). Accordingly, previous studies have shown that a higher reliance on adaptive defenses is associated with lower levels of somatic symptoms (Hyphantis et al., 2013a; Hyphantis et al., 2013b; Waikamp et al., 2021).

As for the Indian sample, mature defense mechanisms did not mediate the association between direct and indirect exposure to ACEs and somatization symptoms. Since the level of exposure to ACE was lower in the Indian sample, a marked regression to immature defense mechanisms was not observed, and therefore the levels of mature defense mechanisms among these participants may not have varied greatly. Consequently, this can potentially be why these mechanisms did not mediate the association between ACE exposure and somatic symptoms, contrary to what was observed in the Faroese sample. In addition, previous studies that reported mediation of defense mechanisms were from samples of adults (Finzi-Dottan & Karu, 2006; Nickel and Egle, 2006). The present study is the first to test defense mechanisms as a mediator between ACE and somatization symptoms among Faroese and Indian adolescents.

Our fourth hypothesis was that dissociation symptoms would mediate the association between direct and indirect exposure to ACEs and somatization symptoms. The results presented

in the empirical article of this dissertation indicated that this hypothesis was fully supported. Specifically, our results indicated that higher direct and indirect exposure to ACE were associated with higher levels of dissociation symptoms, which were then associated with higher levels of somatization symptoms. Our results are in accordance with previous studies on the relationship between these variables, which has shown that memories of the traumatic experiences are dissociated from consciousness as a coping response to deal with the exposure to painful experiences, but later manifest in somatic symptoms (Chu & Dill, 1990; Salmon et. al, 2003; Saxe et al., 1994).

Our fifth and final hypothesis was that serial multiple mediation models would indicate that direct and indirect exposure to ACE were associated with higher levels of immature defense mechanisms and lower levels of mature defense mechanisms, that were relate to higher levels of dissociation symptoms, which were then related to higher levels of somatization symptoms. The results presented in the empirical article of this dissertation indicated that this hypothesis was partially supported.

In the Faroese sample, results revealed that both direct and indirect exposure to ACE were associated with higher levels of immature defense mechanisms, which were associated with higher levels of dissociation symptoms, which were then associated with higher levels of somatization symptoms. This suggests that exposure to ACE can lead to a regression of defense mechanisms employed by Faroese participants, leading them to rely on immature defense mechanisms, which subsequently results in higher dissociation symptoms and, ultimately, higher somatic symptoms. Probably, immature defense mechanisms hinder the processing and integration of ACE, which may result in their dissociation from conscious awareness. Consequently, trauma-related memories are stored at an implicit level, without a verbal component, which makes it difficult for individuals to disclose their traumatic experiences. Since they cannot be symbolized and communicated, individuals can experience bodily sensations associated with these memories. As a result, somatic symptoms may arise or worsen as an unconscious representation of these traumatic experiences (Saxe et al., 1994; van der Kolk & Fisler, 1995).

In the Indian sample, results demonstrated that only direct exposure to ACE was related with higher levels of both immature and neurotic defense mechanisms, which were associated with higher levels of dissociation symptoms, which were then associated with higher levels of somatization symptoms. Among the Indian participants, it is possible that the repetitive nature of direct exposure to ACE results in a regression to more primitive defense mechanisms, leading to higherlevels of dissociation and, in turn, higher somatization symptoms. It is possible to suggest that indirect exposure to ACE is considered normal in the Indian cultural context, unlike in the Faroese context, explaining the non-significant effect on the other variables under study. Additionally, although mature defense mechanisms were not mediators, they were a negative associated with both dissociation and somatization symptoms. These findings suggest that mature defense mechanisms, which allow more conscious awareness of stressors, played a protective role in the face of traumatic events among this sample (Perry et al., 2015; Vaillant, 2011).

The current results obtained in the empirical article presented in this dissertation provide a significant contribution to previous knowledge concerning vulnerability and protective factors associated with the effects of the exposure to multiple ACE on adolescents' mental health, specifically Faroese and Indian adolescents. The present study addresses a gap in research by highlighting the mediating role of defense mechanisms and dissociation symptoms in the association between polytraumatization and somatization. The study also explores their impact on the development and maintenance of trauma-related psychological symptoms, particularly somatization symptoms. Additionally, our results demonstrated that the association between the variables under study was different for Faroese and Indian adolescents. To the best of our knowledge, no previous study has examined the association between these variables and this mediation model following polytraumatization in a sample of adolescents from the Faroe Islands and India.

Limitations

The present study had the following limitations. First, the cross-sectional design disallows causal inferences. It is not possible to infer causality from the serial mediation analyses. Future studies should analyze the relationship between the variables using a longitudinal design.

Second, since the Indian sample is not representative, the current study cannot make general conclusions about Indian adolescents. Third, all data were collected using self-report measures. These measures rely on participants' responses, making them susceptible to subjective interpretations and the influence of social desirability bias. Fourth, ACE report was retrospective, which may be subject to memory bias. Fifth, the study design did not allow to identify if an event had reoccurred. Therefore, a distinction between the effects of a single traumatic event or repeated traumatic events could not be made. Sixth, the traumatic life-event questionnaire has not yet been validated for the samples' population. Furthermore, since it was originally designed to reflect a European sample, certain questions might not fully capture the complexities of the context relevant to this study's participants, specifically in the Indian sample. In addition, it is crucial to consider that cultural differences in the interpretation of the psychological constructs being assessed could have influenced participants' responses to the questionnaire. Seventh, the TSC was primarily employed to assess somatization symptoms in this study. Future studies should be conducted using measures of DSM-5 criteria. Eighth, the data were collected in 2012, implying that there could be differences in the current context of both countries, along with variations in the levels of exposure to ACE among the participants. Given the limitations identified in this study, our findings need to be interpreted with caution and their generalizability should be tested by means of prospective studies. Furthermore, it would be valuable to replicate the mediation hypothesis in adolescents from different cultures.

Implications for research

Future studies on polytraumatization in children and adolescents are required. Furthermore, studies should attempt to replicate the mediation hypothesis with other samples of adolescents, from different cultures. In this sense, the pattern of associations reported in the present study should also be revalidated in future research. Studies conducted to address our hypotheses should contemplate using different instruments. For instance, defense mechanisms could be more effectively assessed by projective testing or dynamic interviews, since they are unconscious psychological processes and participants are mostly unaware of their usage. Additionally, to gain a comprehensive assessment of dissociation symptoms, an instrument that assesses both psychoform and somatoform dissociation symptoms should also be considered. Regarding somatization symptoms, a clinical interview following DSM-5 criteria should be

conducted, in order to properly identify and assess these symptoms. Finally, a longitudinal design could be considered to provide insights into the long-term effects of polytraumatization, contributing to a more comprehensive understanding of the complex interplay between the variables under study. A longitudinal design also enables researchers to examine how the impact of polytraumatization may vary across different developmental stages and allows to assess the effectiveness of possible interventions on mental health outcomes.

Other variables could also be interesting to study in this context. For instance, traumatic experiences have been associated with the development of alexithymia, a condition marked by difficulties in identifying and describing feelings (Koiman et al., 2004; Taylor & Bagby, 2013). Individuals with alexithymia also struggle to differentiate feelings from body sensations, leading them to attribute physical sensations caused by emotional arousal to somatic complaints (Adamowicz et al., 2022). Furthermore, there is evidence linking alexithymia and dissociation, as well as maladaptive defense styles (Grabe et al., 2000; Helmes et al., 2008). As such, alexithymia may act as another mediator or a moderator in future studies. Moreover, it is also known that children exposed to traumatic events are especially at risk for developing insecure and disorganized attachments, which in turn are also associated with various mental health symptoms, including somatic symptoms (Pinto et al., 2021; Waldinger et al, 2006). Therefore, attachment may serve as another underlying mechanism through which polytraumatization and somatic symptoms are linked. Additionally, one variable that could also be interesting to include in future studies is social support. Social support in adolescence encompasses relationships with the parents but also with peers, teachers, extended family and neighbors, and is believed to play a crucial role in the overall well-being of youth exposed to adversity (Bal et al., 2003).

Clinical implications

The findings from this dissertation offer valuable insights for intervening with these adolescents in distinct areas, namely, community intervention, psychological assessment, and psychotherapeutic intervention.

Community Intervention

Trauma-informed interventions for children have traditionally been designed to address specific traumatic experiences, often neglecting the reality of polytraumatized children who have been exposed to multiple forms of trauma. In response to this gap, there is a growing recognition of the need for interventions that are tailored to the complex needs of these children (Banyard et al., 2013; Cohen et al., 2006). In addition to targeting the various traumatic events they have experienced, interventions should also consider the different settings in which children can find themselves exposed to such events. Community interventions can play a crucial role in providing support, resources, and preventive measures to these children. Firstly, implementing community-wide programs that provide information and education about polytraumatization and its effects on children's well-being are important. These programs can help raise awareness, reduce stigma, and promote early identification and intervention. In turn, interventive measures should focus on promoting competencies that may prevent future victimization and distress and assist in building social support networks. Interventions should also consider mental health symptoms that arise from victimization (Berkowitz, 2003; Cyr et al., 2012). The results from our study suggest that polytraumatization increases the risk of mental health problems among adolescents, namely, somatization symptoms. Given that many individuals with somatic complaints seek primary healthcare, it is advisable for healthcare providers to assess trauma history and consider referral to mental health services when necessary. These services should be ensured by the community, linking children to accessible mental health care, child protective services and other social resources. This integrated approach not only supports the children but also has the potential to reduce healthcare costs.

Psychological Assessment

Our study indicated that indirect exposure to ACE was directly and positively associated with somatic symptoms, among Indian participants. This finding provides crucial information for clinicians working with children and adolescents, emphasizing the importance of conducting a comprehensive trauma history evaluation as part of the psychological assessment. Therefore, direct and indirect exposure to traumatic events should be considered, as both relate to psychological symptoms, such as somatization. Moreover, it's essential to consider not only

violent traumatic events, but also other distressing life circumstances that can also be potentially traumatic.

Furthermore, our findings demonstrated the joint mediation of defense mechanisms and dissociation on the association between exposure to ACE and somatization symptoms. In this sense, both defense mechanisms and dissociation assessment should be conducted, since they are psychological processes associated with both exposure to traumatic experiences and to somatic symptoms. In addition, dissociation was a mediator of the association between exposure to ACE and somatization symptoms on its own, appearing to be a key component in this relationship. Given that trauma-related dissociation is often linked with worse psychiatric symptoms, including somatization, dissociation assessment is even more relevant following trauma exposure. Such assessments can help to identify at-risk individuals, which may have a clinical need for tailored early interventions.

Psychotherapeutic Intervention

In the current study, we identified defense mechanisms and dissociation symptoms as underlying psychological mechanisms in the association between polytraumatization and somatization symptoms. These findings suggest that mental health professionals should pay attention to the use of defense mechanisms, especially immature defenses, and dissociation symptoms among adolescents following multiple exposure to ACE, since these clinical manifestations seem to increase the risk to develop somatic symptoms. Therefore, it is recommended that clinicians focus on addressing defenses that restrict awareness of thoughts, emotions, and behaviors, as well as the disruption and/or discontinuity of traumatic experiences in adolescents exposed to adversity.

Psychoanalytically oriented treatment may be indicated for addressing these mental processes, specifically focusing on changing patterns of maladaptive defense use and decreasing dissociative symptoms. A therapeutic approach could involve mentalization-based treatment that centers on increasing mentalization. Mentalization is a capacity that develops in children in the context of safe and secure relationships with their caregivers. Thus, the experience of maltreatment and other distressing events can affect the development of a child's mentalization capacity, which, in turn, is associated with poor mental health outcomes and interpersonal

difficulties (Fonagy & Allison, 2015). For instance, mentalization deficits have been associated with immature defense mechanisms usage (Tanzilli et al., 2021). Likewise, mentalization difficulties following traumatic experiences seem to contribute to higher levels of dissociation and somatization (Ensink et al., 2017; Wagner-Skacel et al., 2022). Therefore, treatment of individuals exposed to ACE with a focus on fostering the development of mentalization could help individuals to rely on more adaptive defenses and prevent symptom formation.

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